COMMENTS ON THE PATHOLOGY AND BACTERI-
OLOGY OF FATAL INFLUENZA CASES, AS
OBSERVED AT CAMP DEVENS, MASS.

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Death from influenza means death from lung complica-[104] tions—pneumonia in some form. The pathological picture of the lungs as seen in a series of postmortems is a kaleidoscopic one. At first it was most difficult to correlate the various gross findings, and it also has been difficult to correlate the microscopic findings, and this part of the work is not yet completed. I have had the advantage of comparing my observations with those of Dr. Goodpasture at the Naval Hospital at Chelsea, Mass., and again with a study of the series of cases we have had at the Peter Bent Brigham Hospital in Boston, and it now is possible to give a fair account of the probable sequence of events and to explain the apparent great differences in the pathology.

In a series such as is presented here two types of lungs stand out as strikingly characteristic findings in this disease. The first is encountered in those cases in which death has occurred within a few days after the onset of pulmonary signs. These cases yield lungs which are partially collapsed, dark red, lax, but meaty in consistency. The pleural surfaces are often partly covered with a dusky red mottling, due to small extravasations of blood beneath the serous coat. There may be a thin layer of dusky red fibrinous exudate upon the pleural surfaces, particularly over the posterior borders. On section these lungs are dark red and wet. They are dripping wet, and the fluid from some portions is a blood-tinged serous liquid and from others dark red and bloody. On close inspection the cut surfaces are usually found to be thickly sprinkled with air vesicles of considerable size. The lung tissue as a whole, after the liquid has drained from it, is brownish-red in color, and somewhat translucent and friable. The mucosa of the bronchi

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[104] is usually very dark red in color, and the bronchial lymph nodes are enlarged and deep red in color.

The other type of lung, which is found in patients that have lived for 10 days or more after the onset of the disease, while showing traces of the type of lesion just described, is characterized by a very extensive bronchitis, with broncho-pneumonia, discrete or confluent, and peri-bronchitis. These lungs are more voluminous than the preceding, but they do not fill the chest cavity at postmortem. They are nodular, and the pleural surfaces occasionally show a striking tracery, due to the injection of the sub-pleural lymphatics. Portions of the surfaces of the lungs may be covered with a thin layer of fibrinous exudate. On section the most prominent feature is the extensive injection of the bronchi, particularly the smaller ones, with a fibrino-purulent exudate. The injection of the bronchi may be so extensive and uniform as to produce geometrical patterns, which are very striking when the condition is accompanied, as it usually is, by a marked infiltration of the inter-lobular septa. A casual inspection suffices to show that the smaller bronchi are distended, usually markedly dilated, and in cases of two weeks' duration spherical and cylindrical bronchiectases are very common. The gross appearances of this type of lung are very much like those described by Dr. Mac-Callum in pneumonias after measles. The condition in fact [105] is one of pan-bronchitis; peri-bronchitis with extensive infiltration of the interlobular septa; and organization in alveoli and bronchioles.

These two predominating types on first consideration seem to represent different processes. I hope to show convincingly that they simply represent different stages of the same process. Before I undertook the study of the Camp Devens cases I had some experience with influenza pneumonias at the Peter Bent Brigham Hospital, and I have since quickly surveyed the material obtained there during the time I was at Camp Devens. The Brigham Hospital cases, on the whole, have been quite different in their gross appearances. On the other hand, the cases at the Naval Hospital in Chelsea have been very similar to those at Camp Devens, and I think a cross-section of the study of one series of cases is very much like that of the other—

to which Dr. Goodpasture agrees. There are many other interesting features of the pathology of the lungs, such as the rapidity with which bronchiectasis occurs and the large number of cases which develop interstitial emphysema, and of course the consequences of both of these conditions; bronchiectatic abscesses and gangrene of lungs from the first, emphysema of the mediastinum and subcutaneous emphysema from the second. Of this series of 28 postmortems, done between the 2d and 32d day of the disease, there were six that showed subcutaneous emphysema; eleven showed emphysema of the mediastinum.

In comparing notes with other pathologists one is struck by the differences in gross appearances of the lungs in different localities. The same is true in regard to the bacteriology. The table which accompanies this report shows the very high percentage of pure *Bacillus influenzæ* pneumonias at Camp Devens; again, a similarity with the series at the Chelsea Naval Hospital as reported by Keegan. However, in the Camp Devens series there were a few cases in which the hemolytic streptococcus and the pneumococcus were found, and these lungs presented different gross appearances. In a cursory analysis of the Brigham Hospital cases autopsied (about 30), I find a predominance of hemolytic streptococcus and pneumococcus lungs. Without going into corroborative details at this time, I may say that in the gross appearance of the lungs I have laid emphasis upon, *B. influenzæ* was the only organism which could be cultivated and I unhesitatingly associate these distinctive conditions with that organism. In lungs showing other types of solidification, other organisms were responsible for the exudation characterizing the pneumonias. The hemolytic streptococcus, the staphylococcus and the pneumococcus, each produces its distinctive picture, the last often that of lobar pneumonia.

While the bacteriological evidence, based upon the assumption that *B. influenzæ* is the cause of influenza, is very good in support of the stand that there is a distinctive lung lesion in these influenza pneumonias, the histological study has afforded very definite proof. Early in this study of the Camp Devens cases, I recognized the fact, as have others, that

[105] a striking type of reaction was present, a condition of acute alveolar emphysema with the deposit of a hyaline fibrinous material on the alveolar walls. The intervening alveoli are compressed and filled with exudate, which in the early cases is largely serous or bloody, containing but little fibrin. It is this acute alveolar emphysema, with the serous and hemorrhagic exudate, that gives the characteristic gross appearance to the lungs in the early stage of the disease. In order to determine how common this lesion is, I have gone over all of the Brigham Hospital autopsies on influenza cases, and find it to be constant. It may be masked by a pneumococcus or streptococcus exudation or by extensive hemorrhage, but its presence can always be determined by the finding of the hyaline fibrin outlining greatly distended air spaces in the lungs. It is the one distinctive feature in the pathology of influenza pneumonias, and its constant occurrence is indicative of the entity of the initial lung infection. The interpretation of this lesion was not easy. The hyaline fibrin, because of its prominence and the juxtaposition of cellular exudate, often simulates the outlines of alveoli. As a matter of fact, it outlines cavities filled with air, which may or may not completely fill groups of alveoli. Although alveolar walls in contact with this fibrin may be necrotic, tissue elements play no part in its formation. A similar hyaline fibrin was found in two cases of emphysema of the mediastinum where the mediastinal areolar tissues were infected by pneumococcus, secondary to pneumococcus pericarditis. The physical characteristics of this fibrin are determined by its contact with air, and an important factor is probably the mechanical compression of strands of fibrin by air. What is the source of the exudation in the alveoli in these early pneumonias? The exudation may be present in alveoli with intact walls, or walls showing very slight reaction, mainly evidenced by activity of the respiratory epithelium. In all cases severe lesions were found in the finest bronchioles, and in the alveolar ducts. The latter show an exudation composed mainly of polymorphonuclear leucocytes and small quantities of fibrin. The walls are filled with leucocytes, and are often necrotic in places. The intralobular bronchioles show severe lesions of the mucosa, and it is often possible to demonstrate

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the source of hemorrhages from capillaries. The obvious explanation, and indeed the only possible one from the material at hand, is that the major injury is to the bronchial system, and mainly in the finest bronchioles and alveolar ducts. To secure the degree of emphysema present it is necessary to assume a valve action of the exudate in the bronchi. The character of the hyaline fibrin deposit around air vesicles and upon the alveolar walls suggests a pouring of exudation into the alveoli from the bronchioles and alveolar ducts, at a time when air is able to pass. Thus the patient is virtually blowing bubbles in his own lungs, into a medium of exudation relatively poor in fibrin.

The mechanism of interstitial emphysema formation is easily seen, where the greatly distended alveoli are in contact with the pleura of interlobular septa. In these locations it is possible to demonstrate rupture of the alveolar walls and the direct continuity of fibrinous strands, partially filling clefts dissected by the air from alveoli to pleural or interlobular connective tissue. A series of gross sections and microscopic sections from lungs with interstitial emphysema shows that the air finds the easiest route of exit from the lung in the connective tissue surrounding blood-vessels. It dissects along [106] blood-vessels to the hilus of the lung and from there along the great vessels and bronchi into the mediastinum, over the pericardium into the anterior mediastinum, and upwards along the trachea into the tissues of the neck, whence it escapes into the subcutaneous tissues. This subcutaneous emphysema may appear very early, as will be seen by the accompanying chart. The earliest case was seven days from the first symptom, which means, of course, a shorter duration of the lung involvement. The majority of the cases were noted on or after the 10th day from the initial symptoms of the disease.

It must be borne in mind in considering the pathology of these lungs, that the lesions are not uniformly distributed, and therefore very extensive injury in portions of one or several lobes are compatible with life for a considerable period of time. The bronchial lesions apparently progress, and may extend throughout the whole of one or both lungs, producing the anatomical picture of the more chronic cases, that of a pan-

[106] bronchitis with bronchiectases and peri-bronchitis. During this period of extension in bronchi, a number of things may happen to the portions of the lungs first involved. They may become secondarily infected with pneumococcus or streptococcus, or the Gram-negative diplococcus called by English workers " Diplococcus mucosus." In rare instances Staphylococcus and Friedlander's bacillus have been encountered. The fate of the tissue depends on the nature of the infecting organism; as, for example, fibrinous exudation with the pneumococcus and abscess formation with the staphylococcus. In a number of instances these portions of the lungs, severely damaged at the onset, did not become secondarily infected; at least, these lungs have shown only the influenza bacillus at the autopsy, and have undergone extensive organization resulting in cicatrices of large sizes. If we take a series of lungs which have shown only the influenza bacillus in cultures and in sections, we may still have all the stages described exclusive of those with secondary infection, and accordingly we must conclude that the reaction to the influenza bacillus is less intense in the later stages of lung involvement than in the earlier. This is shown best in comparing two lungs from the same case, where in one lung, usually the right, we find the severe damage of the early lesion with bronchiectasis and peri-bronchitis, and in the other lung a much less intense bronchial reaction, with much less marked peri-bronchitis, or none at all. The involvement of the pleura in lungs infected solely with the influenza bacillus is very slight. There are hemorrhages into the pleura and perhaps a thin layer of fibrin upon the surface. The amount of fluid in the pleural cavities was always small, though blood-tinged. Empyema was found in cases secondarily infected with the streptococcus or pneumococcus. The involvement of the pleura may result from the extension of the inflammatory process along the interlobular septa and lymphatics, or, and this I believe is more commonly the case, from bronchiectatic cavities situated close to the pleural surface.

Gangrene of the lung was noted in one of the Camp Devens series in a case showing very extensive bronchiectases, with bronchiectatic abscesses. Extensive necrosis of the lung has

been observed in a number of cases in this same series and at the Brigham Hospital—necrosis due to organisms other than the influenza bacillus.

Organization in the pure *B. influenzæ* cases was a common end result. The organization of the exudate begins early, certainly before the 10th day of the disease, and a prominent factor in bringing about this result is, I believe, the plugging of the bronchi with exudation. In patients who had survived three weeks or more there were very complicated gross appearances, due to extensive cicatrization of large portions of the lung. The contraction of interlobular septa, due to the avascular organization of exudate, causes marked distortion of the lobules of the lung, and peculiar lines of retraction on the pleural surfaces.

It is not the purpose of the present report to include the whole pathology of influenza. There are a few interesting features in other organs which are worthy of emphasis, however. Eight of the Camp Devens series showed waxy degeneration of the rectus muscles, and subsequent experience at the Brigham Hospital indicates that it was probably overlooked in some of the earlier postmortems done at Camp Devens. A number of these cases showed rupture and extensive hemorrhage into the rectus muscle. This lesion has been noted in other muscles; for instance, the transversalis, the internal and external oblique muscles, the latissimus dorsi, the pectoralis major and the intercostal muscles. The testes occasionally showed minute petechiæ, but on the whole no striking gross change was observed. Microscopically very striking changes were encountered in nearly every case, namely, the cessation of activity in the seminiferous tubules; actual degenerative changes were frequently noted, and in late cases beginning fibrous tissue replacement of the degenerated tubules. This lesion of the testes seems to be wholly a toxic one, as there is very little cellular reaction. It is difficult to understand why such severe toxic lesions of the muscle and testes should occur, in the absence of effects attributable to toxins in other organs. For instance, the reaction of the spleen is very slight, the heart muscle rarely has shown any gross or microscopic lesion, and in general seems to escape entirely the toxic effect of the dis-

[106] ease. Lesions of the adrenal, when extensive, such as hemor-
rhage, can be attributed to secondary infection, usually the
hemolytic streptococcus. Minor acute lesions are constantly
found in the cortex in influenza cases, but these lesions are
similar to those found in many infectious diseases—the dis-
appearance of lipoid content, and focal necrosis with mono-
nuclear phagocytic cell reaction. The head was opened in
20 of these cases. Infection of the middle ears was found
in 13. Infection of the sphenoidal sinus in 20, frontal sinus
in seven, and of the ethmoidal cells in eight cases. The
bacteriology of the sinuses is given in the chart. Three cases
showed punctate hemorrhages in the cerebral cortex.

BACTERIOLOGY

I do not intend to discuss at length the bacteriology of the
epidemic. The table is a true account of the findings, and I
prefer to have individuals draw their own conclusions. The
opportunities for bacteriological work were particularly good,
[109] as the postmortems were done within a few hours after death
and the cultures were made and studied by myself. The
bacteriological findings were further controlled by staining
sections of the lungs for bacteria, the method employed being
that of Giemsa. Because of their small size the influenza
bacilli are easily recognized. It is interesting to note that in
some early cases the bacilli were found not only in the bronchial
exudate, but in the submucosa of bronchi and in the alveolar
walls of the lung. Inspection of the chart will show that the
influenza bacillus was found in pure culture in one or more
lobes in nine of the 23 cases from which cultures were made.
In sections of lungs from cases in which no cultures were
made, influenza bacilli were found apparently pure in two
cases, and mixed with other organisms in one case. In one
case no influenza bacilli could be found. There were two cases
of lobar pneumonia and one case with gangrene of the lung in
which no influenza bacilli were found. Of 28 cases by cultural
and histological methods combined, *B. influenzæ* was demon-
strated in 23 cases, and in 14 of these in pure culture. It is
worthy of note that the bacilli were present in pure culture in

a number of the late cases. In a number of cases in which [109] influenza bacilli were not found in the lungs by culture, they were found in cultures from the sinuses of the skull or from the middle ear.

An analysis of the table of bacteriological results shows that the bacteriology of the lungs was mixed in a significant number of cases, but it also shows that the one organism occurring with greatest constancy, and in practically every case, was the influenza bacillus. We may regard the pneumococcus, streptococcus, pneumobacillus, and the various micrococci encountered, as secondary invaders, without reasonable doubt. But are we justified in so regarding the influenza bacillus? It is extremely difficult to account for the epidemiological features of this pandemic if we accept the bacillus influenza as the cause. Our lack of knowledge of the pathogenicity of the influenza bacillus and our failure to reproduce the disease in man and animals with pure cultures is also a strong argument against its being the cause of influenza. Yet, on the other hand, it is almost as difficult to explain the constant occurrence of the influenza bacillus in a series such as I have studied. One must keep in mind that our means of identifying the influenza bacillus are meager and that so far but few diagnostic criteria are available; and, by analogy, it seems almost certain that a group of organisms may exist having similar cultural and morphological properties, as is the case with the pneumococci.

The pathology of the lungs indicates clearly that we are dealing with a specific infection with a distinctive pathology in its early stages. The occurrence of *B. influenzæ* in pure cultures in the early stages is a fact of importance in the consideration of the etiology of influenza and I believe firmly establishes the existence of a *B. influenzæ* pneumonia.

CAMP DEVENS AUTOPSIES

No.	Duration	Type of lung	Remarks	Bacteriology	Sections
201	2 days.	Edema and congestion.	Purpuric rash.	Lung = No growth.	Skin = Streptococci in vessel thrombi. Lung = Streptococci in polys in bronchi.
183 **	7 days.	Lobar (?) Bloody. Bilat. solidification.	Subcut. emphysema. No head.	Lung = Pure *B. influenzæ*.	*B. influenzæ* only in bronchi and alveoli.
203 *	7 days.	Bilat. lobar.	Waxy deg. rectus abd. Sph.} Eth.} Sinusitis.	L. L. = *B. influenzæ* + hem. strep. R. U. = *B. influenzæ* + hem. strep. R. L. = *B. influenzæ* + hem. strep. Sph. Sinus = *B. influenzæ*. Pneumococcus IV.	Streptococcus.
207	8 days.	Lobar—left lower.	Fibrinous pleuritis. Fibrinous pericarditis. No head.	L. L. = Pneumococcus II. R. L. = Pneumococcus II.	Pneumococcus.
216	8 days.	Broncho-pneumonia, wet—bloody.	Waxy deg. rectus abd. Sph. Sinusitis.	L. U.} L. L.} R. U.} R. L.} No growth.	Large gram-positive cocci.
188	9 days.	Bilat. lobar.	No head.	Lung = Pneumococcus.	Pneumococcus.
197 *	9 days.	Bilat. broncho-pneumonia, confluent on right. Bronchiectasis.	Sph. Sinusitis.	L. L. = *B. influenzæ* + pneumococcus. R. M. = *B. influenzæ* + pneumococcus. R. L. = *B. influenzæ* + pneumococcus. Sph. sin. = *B. influenzæ* + pneumococcus.	*B. influenzæ*. Pneumococcus. Flat diplococci, Gram-negative.
192 **	10 days.	Bilat. confl. broncho-pneumonia. Bronchiectasis.	Emphysema of media. Empyema, right 400 c. c. No head.	L. L. = *B. influenzæ*. R. U. = *B. influenzæ*. R. L. = *B. influenzæ* + pneumococcus.	L. L.} R. U.} *B. influenzæ*. L. U.} R. L. = *B. influenzæ* + pneumococcus.

195 * *	10 days.	Bilat. broncho-pneumonia. Confl. on left lower. Bronchiectasis.	Bilat. otitis media. Sph.] Eth.] Sinusitis.	L. L. = B. influenzæ. R. L. = B. influenzæ. Sph. sin = B. influenzæ + pneumococcus IV.	B. influenzæ present. Predominating organism a Gram-negative diplococcus.
202 *	10 days.	Bilat. broncho-pneumonia.	Subcut. emphysema. Focal encephalitis. Otitis media. Sph. sinusitis. Waxy deg. Rectus abd.	L. U. = Staphylococcus aureus. L. L. = {B. influenzæ. Gram-negative diplococcus.} R. U. = As above. R. L. = As above. Sph. sin. = B. influenzæ + pneumococcus. Mid. ear = B. influenzæ + pneumococcus.	B. influenzæ in bronchi. Gram-negative diplococcus found in superimposed exudate upon older broncho-pneumonia.
198 * *	12 days.	Bilat. broncho-pneumonia.	Emphysema of media. Otitis media. Fr.] Eth.] Sph.] Sinusitis. Waxy deg. rectus abd.	L. U. = B. influenzæ. L. L. = B. influenzæ. R. L. = B. influenzæ. Sph. sin. = B. influenzæ. Fr. sin. = B. influenzæ.	B. influenzæ predominates. Gram-negative diplococcus found in some sections.
193 * *	13 days.	Confl. bilat. broncho-pneumonia. Bronchiectasis.	Subcut. emphysema. Fr.] Sph.] Eth.] Sinusitis. Waxy deg. rectus abd.	L. U.] L. L.] R. U.] No growth. Sph. sin. = B. influenzæ + pneumococcus IV.	B. influenzæ in small numbers in two lobes. B. influenzæ + large cocci, in two lobes.
201 * *	13 days.	Bilat. broncho-pneumonia. Bronchiectasis.	Subcut. emphysema. Otitis media-bilat. Sph.] Eth.] Sinusitis. Waxy deg. rectus ab.	L. U.] L. L.] R. U.] R. L.] No growth. Sph. sin. = {B. influenzæ. Hem. strep. Pneumococcus IV.} L. mid. ear = {B. influenzæ. Hem. strep. Pneumococcus.}	B. influenzæ in small numbers in bronchi.

CAMP DEVENS AUTOPSIES.—*Continued*

No.	Duration	Type of lung	Remarks	Bacteriology	Sections
214 *	13 days.	Lobar—rt. lower. Fibr. pur. bronchitis. Bronchiectasis.	Pneumococcus meningitis. Fr. Sph. Eth. } Sinusitis. Bilat. otitis media.	L. L. = { *B. influenzæ.* Pneumococcus. } R. L. = { *B. influenzæ.* Pneumococcus. } Sph. sin = { *B. influenzæ.* Pneumococcus. }	Rt. lower = Pneumococci. L. Lung = { *B. influenzæ*, great numbers in bronchi. Pneumococci in alveoli. }
186 *	14 days.	Bilat. broncho-pneumonia. Bronchiectasis.	No head. Subcut. emphysema.	Cultures lost.	*B. influenzæ.* Streptocci. Large cocci.
215 * *	15 days.	Bilat. confl. broncho-pneumonia. Bronchiectasis.	Otitis media-bilat. Sph. sinusitis.	Lung = No growth. Sph. sin. = *B. influenzæ.* Fr. sin. = *B. influenzæ* + strep.	*B. influenzæ* in great numbers, pure.
211 * *	17 days.	Bilat. broncho-pneumonia. Bronchiectatic abscesses.	Otitis media-bilat. Sph. sinusitis.	L. U. = *B. influenzæ*+pueumococcus. L. L. = *B. influenzæ.* R. U. = *B. influenzæ*+pneumococcus. R. M. L. = *B. influenzæ.* R. L. = *B. influenzæ*+pneumococcus. Sph. sin. = lost.	*B. influenzæ* pure, great numbers except in bronchiectatic abscesses and according to lobes. *B. influenzæ* + pneumococcus in bronchiectatic abscesses.
212 *	20 days.	Bilat. broncho-pneumonia. Gangrene R. L.	Cerebral abscesses. Spleen abscesses. Otitis media-bilat. Fr. Sph. Eth. } Sinusitis.	Fr. sin. = *B. iufluenzæ* + pneumococcus II. R. M. E. = *B. influenzæ* + pneumococcus II.	Bacteria of all sorts.
219 * *	21 days.	Broncho-pneumonia—rt. Bronchiectasis.	Emphysema of media. Empyema-bilat. Veg. endocarditis-mitral. Otitis media-bilat. Sph. sinusitis.	No cultures. 9½ hours postmortem.	*B. influenzæ* in great numbers in alveoli and bronchial exudate. Mixed bacteria in bronchiectatic cavities.

223 **	22 days.	Bilat. broncho-pneumonia. Bronchiectasis.	Fibrinous pleuritis. Chr. otitis media-bilat. Fr.} Sph.} Sinusitis. Eth.} Waxy deg. rectus abd.	No cultures. 12½ hours postmortem.	B. influenzæ in great numbers in alveoli and bronchial exudate. Occasional diplococcus.
226	22 days.	Bilat. broncho-pneumonia, advanced organization. Bronchiectasis.	Emphysema of media. Chr. otitis media-rt. Chr. sph. sinusitis.	No cultures. 13½ hours postmortem.	No bacteria except in granulating walls of bronchi, here pneumococcus.
218 **	23 days.	Organizing broncho pneumonia. Bronchiectasis.	Organizing pleuritis. Sph. Sinusitis.	R. U. = B. influenzæ. L. L. = B. influenzæ. R. L. = B. influenzæ, rare col. L. U. = No growth. Sph. sin. B. influenzæ and pneumococcus.	B. influenzæ present. Pneumococcus predominates.
224 **	26 days.	Organizing bilat. broncho-pneumonia.	Emphysema of media. Chr. otitis media-left. Sph. sinusitis. Waxy deg. rectus abd.	L. U. = B. influenzæ. L. L. = B. influenzæ. R. U. = B. influenzæ. R. L. = B. influenzæ+pneumococcus.	B. influenzæ (one slide).
227 **	30 days.	Organizing bilat. broncho-pneumonia.	Fibrinous pleuritis-rt. Sph.} Fr.} Sinusitis. Otitis media-bilat.	L. L. = Contam. R. U. = B. influenzæ. R. M. = B. influenzæ+pneumococcus. R. L. = B. influenzæ+pneumococcus.	B. influenzæ in bronchi (four slides).
194 *	31 days.	Confl. bilat. broncho-pneumonia. Bronchiectasis.	Otitis media bilat. Sph. sinusitis.	L. L. = Hem. strep. R. L. = B. influenzæ + hem. strep. Sph. sin. = B. influenzæ + strep.	Streptococcus. B. influenzæ (one of seven slides).
213 **	32 days.	Collapsed lungs, negative.	Empyema-bilat. Veg. endocarditis. Waxy deg. rectus abd. No head.	L. L. = B. influenzæ, pure. R. L. = B. influenzæ, pure. Pericard. = pneumococcus II.	B. influenzæ in bronchi and alveoli. Pneumococci in bronchi.
225 *	?	Lobar—L. upper. Gray hepatization.	No head.	No cultures. 12 hours post mortem.	B. influenzæ in bronchial exudate. Pneumococcus.
191	12 days.	Confl. bilat. broncho-pneumonia.	No head. Subcut. emphysema.	Lung = No growth.	B. influenzæ in bronchial exudate. Pneumococcus.

CHART.—The following abbreviations are used:

L. U. = Left upper lobe.	Sph. = Sphenoid.
L. L. = Left lower lobe.	Eth. = Ethmoid.
R. U. = Right upper lobe.	Bilat. = Bilateral.
R. M. = Right middle lobe.	Confl. = Confluent.
R. L. = Right lower lobe.	

The stars in the first column indicate the presence of *B. influenzæ* in the lungs; double stars, in pure culture in one or more lobes.

FIG. 1.—Photograph of a Kaiserling specimen showing acute interstitial emphysema, characteristic of the early lesion in uncomplicated influenza pneumonia. Case 195.

FIG. 2.—Photograph of a Kaiserling, specimen showing uncomplicated influenza pneumonia, of 17 days' duration. Case 211.

FIG. 3.—Photograph of a Kaiserling specimen of uncomplicated influenza pneumonia, of 26 days' duration. Note the extensive cicatrization with bronchiectasis. Case 224.

FIG. 4.—Interstitial emphysema in uncomplicated influenza pneumonia, of 13 days' duration. Case 204.

FIG. 5.—Low power photomicrograph, from an uncomplicated early influenza pneumonia, of 10 days' duration. Case 192.

FIG. 6.—Uncomplicated influenza pneumonia, of 17 days' duration, with extensive organization. Case 211.

FIG. 7.—Clump of influenza bacilli in alveolar exudate, early influenza pneumonia. Case 183.

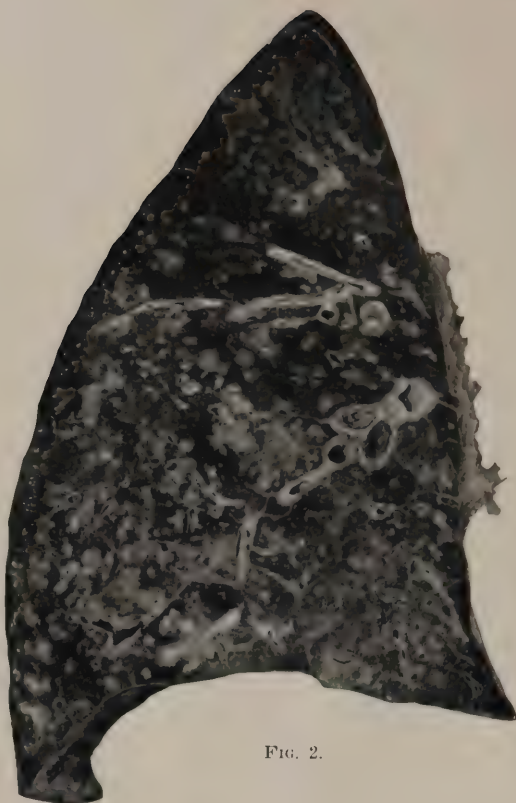FIG. 8.—Influenza bacilli in alveolar walls, early influenza pneumonia. Case 183.
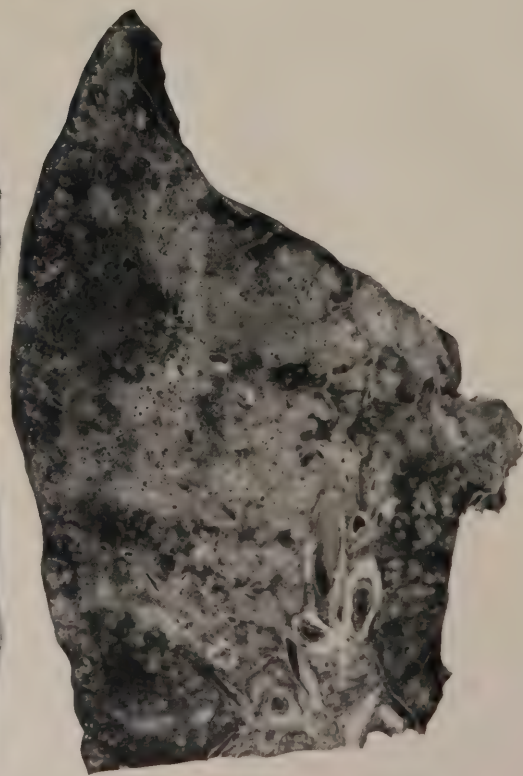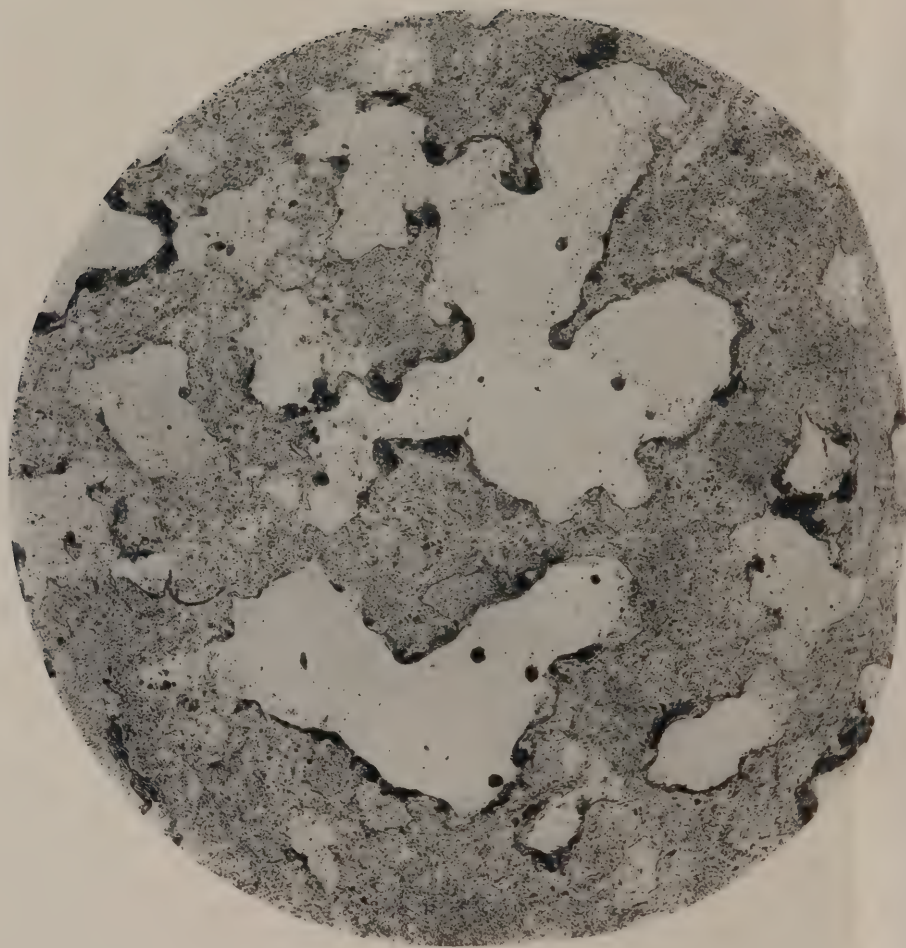
PLATE IX
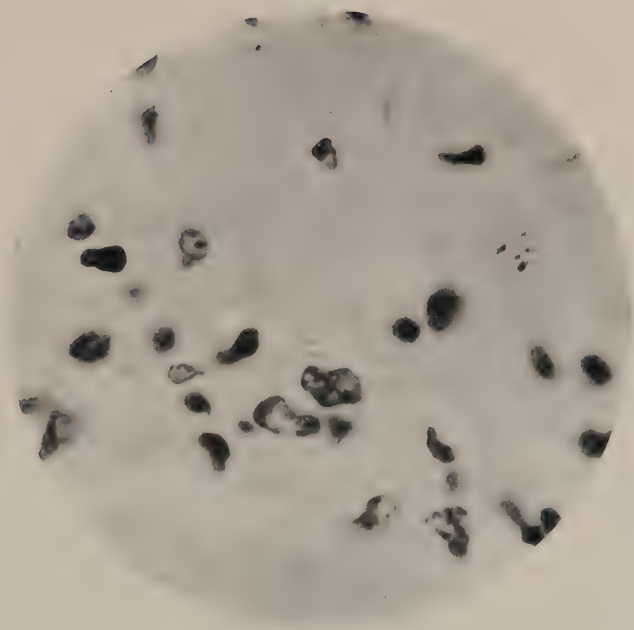

Fig. 1.

Fig. 2.

Fig. 4.

Fig. 3.

Fig. 6.

PLATE X

FIG. 5.

FIG. 8.

FIG. 7.